

Chylothorax and cirrhosis of the liver: A case report

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Abstract

Chylothorax occurring in the setting of a lymphoma or a surgical procedure involving the area around the thoracic duct is a well-known phenomenon. Less common is the occurrence of chylothorax in conjunction with cirrhosis of the liver. Due to the paucity of data, it is uncertain if chylothorax is an associated or an independent sign of cirrhosis. The case reports in the literature favor the former, as demonstrated in this case of a patient with cirrhosis of the liver who developed a chylothorax.

Case Presentation

This is an 81-year-old male with no prior history of significant medical illness or surgery who several months prior to admission presented to his primary care physician's office with mild abdominal distension and abnormal liver function tests. An upper abdominal ultrasound was performed and demonstrated cirrhotic changes of the liver, splenomegaly and a 3.4 cm heterogeneous echogenic solid mass in the right lobe of the liver. Abdominal CT scan confirmed these findings and raised the possibility of a hepatoma. A liver gallium scan revealed no significant uptake of gallium by the liver. Spironolactone decreased the distention and the patient lost weight. A repeat CT scan 3 months later revealed an increase in the size of the liver tumor to 3.7 cm and laparoscopic exploration of the patient's abdomen was recommended. At this time, the patient was still asymptomatic.

On physical examination, the patient had several small spider angiomas on his chest wall but no other stigmata of hepatic dysfunction. On the abdominal exam there was mild distention, a slightly firm, non-tender liver palpable below the costal margin in the midclavicular line and a fluid wave. Trace/1+ edema was noted in the lower extremities. The rest of this patient's physical examination was within normal limits. Alpha-fetoprotein was 4.6 ng, total bilirubin was 0.5 mg/dL, albumin was 2.8 gm/dL, prothrombin time was 13.3 seconds, activated partial thromboplastin time was 29.2 seconds, International Normalized ratio was 1.0, complete blood count and electrolytes were within normal limits.

A laparoscopic exploration was performed and ultrasound guided biopsy taken. Biopsy revealed well-differentiated hepatocellular carcinoma with changes suggestive of cirrhosis. With this finding radiofrequency thermal ablation treatment was performed. Approximately 500 mL of peritoneal fluid was noted during the surgery.

Post-operatively the patient began to complain of shortness of breath, a productive cough, and pain on his right thorax upon inspiration. Crackles were heard in his lungs along with decreased breath sounds and his abdomen was still distended but non-tender. The assessment was post-operative ascites and the patient's spironolactone dosage was increased and respiratory therapy was ordered. The patient's condition did not improve over the next few days and a chest x-ray revealed increased pleural effusion. An ultrasound-guided thoracentesis was performed on the patient's right hemithorax and 1.5 L of dark yellow clear fluid was aspirated. A repeat chest x-ray revealed a significant decrease in pleural effusion and confirmed placement of the internal jugular catheter tip in the proximal superior vena cava. An abdominal CT revealed no ascites. The patient's condition improved.

Approximately 2 days following the thoracentesis, the patient again began to become increasingly short of breath and coughing recurred. Pleural effusion began to reaccumulate in the patient's right pleural space per physical examination and chest x-ray. His abdominal distention increased and he developed a 12-15 cm hydrocele of his scrotum. A second ultrasound-guided thoracentesis was performed on the right side, 2 liters of cloudy fluid were drained, and a chest tube was placed. The placement of the chest tube was assessed radiographically revealing no pneumothorax. Pleural fluid was sent for analysis. The chest tube was kept in place until the fluid stopped draining.

The patient's condition improved dramatically after the second thoracentesis. His activity level increased, he was weaned off of oxygen, his abdominal distention decreased and his hydrocele resolved. The chest tube continued to drain a cloudy, milky fluid. Analysis of this fluid revealed 399 mg/dL triglycerides and no bacterial growth consistent with a chylothorax. Diet was switched initially to fat restriction then to TPN. The color of the drainage changed from milky white to straw-colored and over the next two weeks the volume of fluid drained daily decreased. The chest tube was clamped then removed and on the 31st post-operative day the patient was discharged from the hospital.

Discussion

A chylothorax is diagnosed when the pleural fluid has a triglyceride level greater than 110 mg/dL, a pleural fluid to serum triglyceride ratio higher than 1, and a pleural fluid to serum cholesterol ratio lower than 1. It is often common practice for physicians to rely upon the gross appearance of the pleural fluid to make the diagnosis of hydrothorax or chylothorax, but this may not always hold true. In a series of 24 patients with confirmed chylothorax reported by Romero et al, 14 were milky, 5 were bloody, 3 were turbid yellow, and 2 were clear yellow. Only 58% of the patient's drained the

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classic milky white fluid. Thus the common assumption that a straw-colored pleural fluid is nonchylous may contribute to chylothorax being underreported.

The thoracic duct normally conveys between 1500 and 2500 mL of chyle daily so a leak in the lymphatic system can have major physiologic repercussions. Loss of excess amounts of chyle can lead to malnutrition, water and electrolyte loss, hypolipidemia, and immunosuppression. The causes of chylothorax include tumor (54%), trauma (25%), idiopathic (15%) and miscellaneous (6%). Of tumors, lymphoma is the cause of chylothorax 75% of the time. Traumatic causes include cardiovascular and thoracic surgical procedures, esophageal resections and transhiatal resection. Non-surgical traumatic causes documented in the literature include nonpenetrating trauma in which the spine is hyperextended or a vertebra is fractured after the ingestion of a fatty meal, weight lifting, straining, severe bouts of coughing or vomiting, childbirth, and vigorous stretching while yawning.

Chylothorax is the most common type of neonatal pleural effusion. These chylothoraces may be due to rupture of the thoracic duct from trauma during delivery or to developmental abnormalities of the thoracic duct.

Just as a hydrothorax can arise from ascites a chylothorax may also originate from chylous ascites. Also, chylous ascites may originate from cirrhosis of the liver and this is thought to occur in 6% of patients with cirrhosis. This combination of cirrhosis, chylous ascites and chylothorax is an unusual finding. Its rarity precludes any accurate estimates as to its actual incidence but one author has estimated that up to 1% of chylothorax cases are caused by cirrhosis of the liver. Another author stated that 20% (5 out of 24) of their patients with chylothorax had cirrhosis as the main cause.

The proposed mechanism is that increased portal hypertension secondary to cirrhosis of the liver increases the flow and thus pressure into the hepatic lymphatic system and this pressure is transmitted to the thoracic duct causing transudation of chyle into the peritoneal cavity producing chylous ascites. This was proven in part by Dumont and Mulholland who showed that lymph flow in the thoracic duct is markedly increased in patients with cirrhosis. Alternatively, exceptionally high pressure together with degenerative changes in the splanchnic lymph vessels may lead to rupture of small lymphatics and leakage of whole intestinal lymph into the ascitic fluid. This chylous fluid then enters the pleural cavity via microscopic anatomical defects in the diaphragm. Cirrhotic chylothorax was always a transudate according to Light's criteria and the pleural effusions is almost always on the right side.

In the case presented above the patient had cirrhosis of the liver, pleural effusion and ascites. Thoracentesis was performed twice but only fluid from the second procedure was analyzed. The ascites decreased following aspiration of pleural fluid demonstrating an open communication between the peritoneum and the pleural cavity. As stated above, the gross appearance is not always a reliable indicator of a pleural fluid being chylous or nonchylous. Thus two possibilities exist for this patient. The first is that we assume the patient's pleural effusion was initially chylous even though the first thoracentesis did not reveal fluid of a milky white color. Then the pathogenesis of the chylothorax would be as described above where the increase in pressure in the portal system secondary to cirrhosis of the liver is transferred to the lymphatic system and transudate

from the lymphatic system enters the peritoneal cavity. Since this patient had just received RITA to the liver, the liver capsule and adjacent diaphragm might have undergone inflammatory changes including, increased vessel permeability, further facilitating the transfer of fluid from peritoneum to pleural cavity.

The second possibility is that the patient's original pleural effusion was not chylous and that the chylothorax did not develop until several days into the patient's hospital course. Again, the first tap revealed straw-colored fluid and not milky fluid but in this scenario we are assuming that initially the fluid is nonchylous. Nonchylous ascites is a relatively more common complication of cirrhosis of the liver when compared with chylous ascites. As has been previously demonstrated and is now well known, unidirectional flow of ascites fluid through diaphragmatic defects into the pleural space in patients with cirrhosis can occur causing a hydrothorax in association with cirrhosis. This pleural fluid may have turned chylous after the several days of coughing the patient had after the first thoracentesis. Here although the ascites did not originally contain chyle, chyle may have started to accumulate within the peritoneal cavity when the increased intraabdominal pressure secondary to coughing damaged the lymphatic vessels that were already under increased pressure from the cirrhotic liver.

It is also possible that the chylothorax was secondary to trauma from thoracentesis. Numerous thoracic duct anomalies have been reported and the existence of such an anomaly would certainly increase the chance of a traumatic etiology. Nevertheless, both thoracenteses were performed under ultrasound guidance and as such the combined probability of having an anomaly of the thoracic duct and also damaging it during an ultrasound-guided thoracentesis is highly unlikely.

One last assumption has to be made in order for all of this to be true. The ascites fluid was never analyzed or visualized directly. Yet, we may assume that the ascites fluid was identical to the pleural fluid and that the two were in direct connection with each other since the decrease in ascites was synchronous with the decrease in pleural fluid.

Conclusion

A chylothorax secondary to cirrhosis of the liver is a rare finding. In most instances the diagnosis of chylothorax will be grossly evident, but at other times a high degree of suspicion will be needed. Since the treatment of a chylothorax does differ slightly from the treatment of a hydrothorax an accurate diagnosis is critical. Therefore, it is important for physicians to know that their patients with cirrhosis of the liver and a pleural effusion could possibly have a chylothorax.

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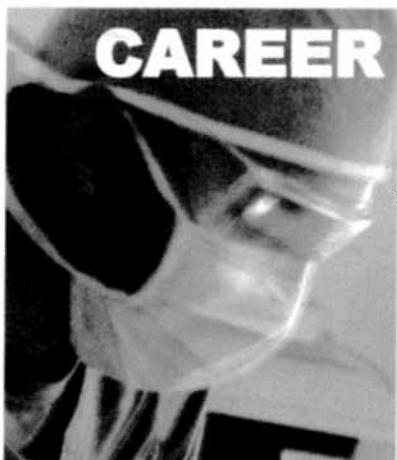
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